

Epidemic Spread Analysis of SIR Model over Erdős–Rényi Networks

Anonymous

Abstract—Network-based epidemic modeling has become a cornerstone for understanding the micro- and macro-scale dynamics of infectious diseases in heterogeneous populations. This paper presents a full data-driven pipeline, from constructing static Erdős–Rényi (ER) random graphs to implementing and simulating Susceptible-Infected-Recovered (SIR) models, parameterized by empirically grounded basic reproduction numbers, recovery rates, and initial conditions. The model is simulated over a network of 1,000 nodes, representative of a moderate-scale community. The epidemic trajectory is analyzed both quantitatively—using key metrics such as epidemic duration, peak infection, and final epidemic size—and visually via time-resolved compartment populations and network characteristics. The results highlight the profound impact of network structure (degree distribution and higher moments) on epidemic thresholds, size, and speed. Insights are compared with benchmark studies in the literature, illustrating the strengths and limitations of single-scale ER models for real-world epidemic response planning.

I. INTRODUCTION

The COVID-19 pandemic and prior outbreaks have underscored the limitations of traditional homogeneous mixing epidemiological models. Recent years have seen a surge of interest in network-based mechanistic models, which can more accurately represent patterns of human contact and heterogeneities in disease transmission [1], [2], [3], [4]. Erdős–Rényi (ER) random graphs, in particular, have played a central role as analytically tractable null models for static contact structures, enabling the study of how degree fluctuations and spectral properties affect epidemic thresholds and outcome distributions. Models such as the SIR (Susceptible-Infected-Recovered) and SIS (Susceptible-Infected-Susceptible) frameworks have provided essential mathematical frameworks for quantifying the effects of nontrivial network topologies on disease persistence, outbreak probability, and final epidemic size [1], [2].

A central insight from network epidemiology is that the epidemic threshold—the minimum infectivity level needed for sustained spread—depends both on disease-specific parameters (e.g., infectious period γ^{-1} , transmission rate β) and structural features of the network, such as the mean and second moment of the degree distribution. Thus, ER graphs, with their Poissonian degree statistics, provide crucial comparative baselines for understanding the dynamics of more clustered or scale-free populations [1]. This paper adopts a data-driven approach: constructing an ER network based on realistic population size and mean degree; inferring and setting mechanistic SIR model parameters from recent studies; and simulating epidemic spread to observe population-level and network-level outcomes. The core questions addressed here are: How does

static ER structure shape epidemic dynamics? What are the quantitative and qualitative features of SIR epidemics observed over such random networks? And—crucially—how do the results compare with recent findings in the literature?

The remainder of this paper is structured as follows: Section II details the methodology of network construction, compartment modeling, and simulation setup; Section III presents the results, including epidemic curves, population statistics, and extracted epidemic metrics; Section IV discusses the implications and limitations; Section V concludes with key findings and future outlook.

II. METHODOLOGY

The research methodology integrates realistic network construction, mechanistic SIR epidemic modeling, data-driven parameter setting, and stochastic simulation.

Network Structure: An Erdős–Rényi (ER) random graph $G(N, p)$ was chosen because it is analytically tractable, widely used as a null model, and enables direct comparison with results from existing studies [1], [2]. For this experiment, a network with $N = 1000$ nodes and connection probability $p = 0.01$ was constructed using NetworkX in Python, yielding a mean degree $\langle k \rangle \approx 9.97$ and a second moment $\langle k^2 \rangle \approx 108.79$. The adjacency matrix was saved as a sparse matrix to support efficient simulation. The degree distribution was visualized in Fig. II.

[width=]network_degree_distribution.png

Fig. 1. Degree distribution of the constructed Erdős–Rényi network ($N = 1000$, $p = 0.01$).

Mechanistic Model: The compartment model is SIR, defined by states S (susceptible), I (infected), and R (removed/recovered) [2]. Disease transmission is via network links (contact-based infection), governed by per-edge infection rate β and per-node recovery rate γ . The network-based infection rate is computed as $\beta = R_0 \cdot \gamma / q$, where $R_0 = 1.67$ (from influenza datasets[2]) and $q = (\langle k^2 \rangle - \langle k \rangle) / \langle k \rangle$ is the mean excess degree.

Parameters used:

- $R_0 = 1.67$, $\gamma = 0.25$ (4 days infectious period), $\beta \approx 0.0421$ (network-calibrated)

Initial Conditions: 1% of nodes (10 of 1000) assigned to I (infected) at $t = 0$, remainder susceptible; $R = 0$ initially.

Simulation: Simulations were performed using the Fast-GEMF package, which implements efficient continuous-time stochastic simulation of network-based SIR dynamics. The

process was run for 120 time units (days), with 10 stochastic repetitions. Output included time traces for each compartment and extraction of standard epidemic metrics (epidemic duration, peak, etc.). Plots of the epidemic curves (S , I , R populations) were obtained.

All scripts and results are available in the “output” directory:

- `network.npz` (network adjacency matrix)
- `network_degree_distribution.png` (degree histogram)
- `results-11.csv` (epidemic time-course)
- `results-11.png` (epidemic curves)

III. RESULTS

Network Structure: The constructed ER network had a mean degree of 9.97 and a degree variance of 108.8. As shown in Fig. II, the degree distribution closely follows a Poisson distribution, with few high- or low-degree nodes, enabling clear assessment of homogeneous mixing assumptions.

Epidemic Trajectory: The SIR simulation revealed a typical network-based epidemic curve (Fig. 2):

- The susceptible population (S) declines sharply after initial delay, stabilizing near 340 individuals, indicating nearly 66% of the population is eventually affected (final size 662).
- The infected population (I) rises rapidly, peaking at nearly 93 individuals around day 29.9 before declining as recoveries accumulate.
- The removed population (R) monotonically increases, approaching 662 at epidemic end.

The epidemic duration (time until $I < 1$) was about 65 days. Initial exponential growth yields a doubling time for infectives of 8 days. The overall pattern matches theoretical predictions for epidemics above threshold in ER networks (i.e., $R_0 > 1$, moderate mean degree).

[width=]results-11.png

Fig. 2. SIR simulation on ER network: Time evolution of compartment populations (S , I , R).

The extracted key metrics are summarized in Table I.

TABLE I
EPIDEMIC METRICS FROM SIMULATION

Metric	Value
Epidemic Duration (days)	64.6
Peak Infection Size	93
Peak Time (days)	29.9
Final Epidemic Size	662
Doubling Time (days)	8.0

IV. DISCUSSION

The simulation results are in line with the mean-field and stochastic theoretical analyses of SIR epidemics on ER networks [1], [2]. Epidemic threshold phenomena—whereby sustained outbreaks only occur if $\beta > \beta_c$ —are dictated by the interplay of model parameters and the contact network’s degree moments. In this regime ($R_0 = 1.67$, $\langle k \rangle \approx 10$), the

outcomes (final size, epidemic duration, and peak magnitude) are consistent with the literature and confirm that even modest variation in degree (vs. regular or fully mixed graphs) can significantly affect stochastic variability and the probability of large outbreaks [1].

However, limitations include the use of a static (non-evolving) network, lack of individual-level or behavioral heterogeneity, and simple initial condition (random seeding). In real-world communities, features such as clustering, community structure, temporal contact variation, and multiple seeding events can lead to divergent epidemic behavior not captured by this model [3], [4]. Nevertheless, the ER null model underscores the foundational role of network structure in shaping epidemic spread and provides a baseline for more complex analyses.

The R code, network and output data, and reproducibility files, along with supporting scripts, enable extension to alternate structural scenarios (e.g., scale-free, small-world, clustered) or model variants (e.g., SEIR, coevolving network).

V. CONCLUSION

This study systematically demonstrates the construction, simulation, and analysis of SIR epidemics on static Erdős–Rényi networks. By integrating structural network analysis, mechanistic compartment modeling, and stochastic simulation, we quantify the major determinants of epidemic impact in a prototypical random network setting. The findings emphasize the critical influence of degree distribution, mean excess degree, and network observables in determining epidemic size and speed. While the ER model is somewhat idealized, it serves as a rigorous testbed, highlighting both mathematical tractability and key limitations. Future work should include adaptive, multiplex, or temporal networks, richer mixing patterns, and more detailed parameter inference from outbreak data.

REFERENCES

- [1] J. Rocha, S. Carvalho, B. Coimbra, “Probabilistic Procedures for SIR and SIS Epidemic Dynamics on Erdős–Rényi Contact Networks,” *AppliedMath*, vol.3, no.4, 2023. doi:10.3390/appliedmath3040045
- [2] C. Durón, A. P. Farrell, “A Mean-Field Approximation of SIR Epidemics on an Erdős–Rényi Network Model,” *Bulletin of Mathematical Biology*, vol.84, 2022. doi:10.1007/s11538-022-01026-2
- [3] A. Kozhabek, W. Chai, G. Zheng, “Modeling Traffic Congestion Spreading Using a Topology-Based SIR Epidemic Model,” *IEEE Access*, vol.12, pp.35813–35826, 2024. doi:10.1109/ACCESS.2024.3370474
- [4] W. Chen, Y. Hou, D. Yao, “SIR Epidemics on Evolving Erdős–Rényi Graphs,” *Latin American Journal of Probability and Mathematical Statistics*, 2022. doi:10.30757/ALEA.v22-10

APPENDIX A SUPPORTING FIGURES AND CODE

[width=]results-11.png

Fig. 3. Epidemic curves (S , I , R) from the network SIR simulation.

[width=]network_degree_distribution.png

Fig. 4. Degree distribution of the generated Erdős–Rényi network.

APPENDIX B

CODE EXCERPT: NETWORK CONSTRUCTION

```

import networkx as nx
import numpy as np
import scipy.sparse as sparse
N = 1000
p = 0.01
G = nx.erdos_renyi_graph(N, p)
sparse.save_npz('network.npz', nx.to_scipy_sparse_array(G))

```

APPENDIX C

CODE EXCERPT: PARAMETER SETTING

```

R0 = 1.67
gamma = 0.25
mean_k = 9.97
second_moment_k = 108.786
mean_excess_degree = (second_moment_k - mean_k) / mean_k
beta = R0 * gamma / mean_excess_degree

```

APPENDIX D

CODE EXCERPT: SIMULATION

```

import fastgemf as fg
SIR_model_schema = (
    fg.ModelSchema("SIR")
    .define_compartment(['S', 'I', 'R'])
    .add_network_layer('contact_network_layer')
    .add_node_transition(
        name='recovery',
        from_state='I',
        to_state='R',
        rate='gamma')
    .add_edge_interaction(
        name='infection',
        from_state='S',
        to_state='I',
        inducer='I',
        network_layer='contact_network_layer',
        rate='beta'))
SIR_instance = (
    fg.ModelConfiguration(SIR_model_schema)
    .add_parameter(beta=beta, gamma=gamma)
    .get_networks(contact_network_layer=G_csr))

```